12-2014

Asymmetrical pedaling patterns in Parkinson's disease patients.

Amanda L. Penko
Stokes Cleveland VA Medical Center

Joshua R. Hirsch
Cleveland Clinic,

Claudia Voelcker-Rehage
Jacobs University,

Philip E. Martin
Iowa State University, pemartin@iastate.edu

Gordon Blackburn
Cleveland Clinic

See next page for additional authors

HHS Public Access

Author manuscript

Clin Biomech (Bristol, Avon). Author manuscript; available in PMC 2015 December 01.

Published in final edited form as:

Clin Biomech (Bristol, Avon). 2014 December ; 29(10): 1089–1094. doi:10.1016/j.clinbiomech.2014.10.006.

Asymmetrical Pedaling Patterns in Parkinson's Disease Patients

Amanda L. Penko[1,3], **Joshua R. Hirsch**[1], **Claudia Voelcker-Rehage**[5], **Philip E. Martin**[6], **Gordon Blackburn**[4], and **Jay L. Alberts**[1,2,3]

[1]Department of Biomedical Engineering, Cleveland Clinic, Cleveland, Ohio, USA.

[2]Center for Neurological Restoration, Cleveland Clinic, Cleveland, Ohio, USA

[3]Cleveland FES Center, L. Stokes Cleveland VA Medical Center, Cleveland, Ohio, USA

[4]Department of Preventive Cardiology, Cleveland Clinic, Cleveland, Ohio, USA

[5]Jacobs University, Bremen, Germany

[6]Iowa State University, Ames, Iowa, USA.

Abstract

Background—Approximately 1.5 million Americans are affected by Parkinson's disease [1] which includes the symptoms of postural instability and gait dysfunction. Currently, clinical evaluations of postural instability and gait dysfunction consist of a subjective rater assessment of gait patterns using items from the Unified Parkinson's Disease Rating Scale, and assessments can be insensitive to the effectiveness of medical interventions. Current research suggests the importance of cycling for Parkinson's disease patients, and while Parkinson's gait has been evaluated in previous studies, little is known about lower extremity control during cycling. The purpose of this study is to examine the lower extremity coordination patterns of Parkinson's patients during cycling.

Methods—Twenty five participants, ages 44-72, with a clinical diagnosis of idiopathic Parkinson's disease participated in an exercise test on a cycle ergometer that was equipped with pedal force measurements. Crank torque, crank angle and power produced by right and left leg were measured throughout the test to calculate Symmetry Index at three stages of exercise (20 Watt, 60 Watt, maximum performance).

Findings—Decreases in Symmetry Index were observed for average power output in Parkinson's patients as workload increased. Maximum power Symmetry Index showed a significant difference in symmetry between performance at both the 20 Watt and 60 Watt stage and the maximal resistance stage. Minimum power Symmetry Index did not show significant differences across the stages of the test. While lower extremity asymmetries were present in Parkinson's patients during

Corresponding Author: Jay L. Alberts, PhD Cleveland Clinic 9500 Euclid Ave./ND20 Cleveland, OH 44195 USA Phone: 216-445-3222 Fax: 216-444-9198 albertj@ccf.org.

pedaling, these asymmetries did not correlate to postural instability and gait dysfunction Unified Parkinson's Disease Rating Scale scores.

Interpretation—This pedaling analysis allows for a more sensitive measure of lower extremity function than the Unified Parkinson's Disease Rating Scale and may help to provide unique insight into current and future lower extremity function.

Keywords

Parkinson's disease; pedaling; postural instability; UPDRS; Exercise test; Posture; Balance; Gait dysfunction

Parkinson's disease (PD) [1] affects nearly 1.5 million Americans with medical treatment costs approaching $25 billion annually. According to current models of basal ganglia function, changes in the pattern and rate of neuronal activity in the basal ganglia result in a decrease in motor cortical activation in PD patients [2]. This decreased motor activation may limit PD patients' ability to generate and coordinate voluntary movements, including gait and postural stability. Postural instability and gait dysfunction are two of the four cardinal motor symptoms of PD which include bradykinesia, resting tremor, rigidity and postural instability and gait dysfunction. Clinical evaluation of postural instability and gait dysfunction involves examining balance and walking patterns through subjective assessment using the Unified Parkinson's Disease Rating Scale (UPDRS) [3, 4]. Postural instability and gait dysfunction is rated in this exam on a scale from 0-4, with higher scores indicating greater deficits. These deficits affect activities of daily living and may necessitate assistive walking devices to compensate for loss of lower extremity and postural control. Despite postural instability and gait dysfunction having significant impact on activities of daily living and quality of life, the UPDRS postural instability and gait dysfunction measures only comprise two out of the eighteen Motor-III assessment questions [5, 6]. This suggests that not all factors of postural instability and gait dysfunction are assessed during the UPDRS exam.

Pedaling, like walking, is a bipedal motor task, with both activities requiring the same principles of lower extremity coordination and rhythmic pattern generation [7-10]. Due to the similarities of these tasks, it is reasonable to suggest that dysfunction or asymmetry present in one of these activities may also be present in the other. Therefore, the quantification of pedaling kinetics, which allows for a more precise measure of lower extremity function than the two assessment questions on the UPDRS, in a clinical population with gait impairments may be useful for clinicians in the determination of postural instability and gait dysfunction. Cycling kinetics, which have not been used traditionally as a clinical diagnostic tool for neurological disorders such as PD, are not well documented in any disease population, despite the fact that cycling is often recommended for rehabilitation or exercise in neurological populations [11-15]. Pedaling kinetics during cycling has been well described for young, healthy populations [16-20]. While this is valuable in efforts to improve cycling efficiency in athletes, this data can also serve as normative data for comparative analysis within clinical populations as well. The identification and characterization of deviations from the typical pedaling motion could be beneficial in assessing lower extremity function in individuals with movement impairments.

Understanding pedaling kinetics in these disease populations may be useful in tracking disease progression, measuring intervention efficacy, or lending insight into specific areas of function to be targeted for intervention. Recently, we have shown that a “forced-exercise” intervention, delivered via stationary tandem bicycle, resulted in a significant improvement in global motor functioning of PD patients [21]. In this initial study, pedaling kinetics was not quantified although it would have been useful to determine lower extremity function and possible alteration.

Lower extremity asymmetry and how it relates to postural instability and gait dysfunction has not yet been studied in a PD population. The primary metric of interest in PD pedaling kinetics is the symmetry of power output during pedaling. During one pedaling revolution, each limb provides a maximal torque at approximately 100 degrees when the leg is pushing on the pedal (downstroke), through which it powers the motion of the bicycle. This maximal torque is contraposed by the opposite leg generating a smaller and negative torque, when the leg is effectively “pulling up” the other pedal (upstroke) (see Figure 1). In order to quantify the pedaling motion, this maximum and minimum generated torques, along with the average torque generated for each revolution, must be measured, and the outputs from each limb during the same revolution are then able to be compared using a Symmetry Index (SI). These SI values provide insight into the pedaling coordination of the maximum and minimum outputs and of the average level of function during each revolution.

Power output of the lower extremity, which is directly related to crank torque, is an indicator of cycling performance [22]. This relationship allows for identification of lower extremity asymmetries through examination of crank torque produced during pedaling. Healthy individuals, including both recreational and competitive cyclists, exhibit some degree of interlimb asymmetry, ranging from five to twenty percent, during pedaling [23] with the dominant leg applying greater torque. Though these asymmetries are present, studies have shown that they become less pronounced as pedaling rate increases from 60 rpm to 80 rpm [23], from 40 rpm to 100 rpm [24], and from 60 rpm to 120 rpm [25]. These previous studies suggest that while some low extremity asymmetries may be the norm, they are not great enough to affect normal daily activity.

Despite the prevalence of pedaling kinetics characterization in healthy adults, pedaling kinetics in clinical populations such as PD has received relatively little attention. The aim of this project was two-fold: 1) characterize the pedaling kinetics in PD patients during a maximal effort graded exercise test (GXT); and, 2) assess the relationship between measures of pedaling kinetics and clinical measures of gait and postural stability. It was hypothesized that pedaling asymmetries would be found in PD patients, to a similar degree as found in healthy populations, and that these asymmetries would show positive association with clinical measures of gait and postural stability, with worsening clinical ratings corresponding to increased cycling asymmetry.

METHODS

Participants

Twenty five participants, (males n=11 ; females n=14) ranging from 44-72 years of age, with a clinical diagnosis of idiopathic Parkinson's disease and who were Hoehn and Yahr stage II-III when off anti- parkinsonian medication were eligible to participate (see Table 1 for demographics). Individuals with existing cardiopulmonary disease, stroke, dementia, diabetes mellitus, or any medical or musculoskeletal contraindication to exercise were excluded. Evaluation of inclusion criteria was made based on the patient's medical history and physical examination. All participants were recruited from the Cleveland Clinic or local neurology offices and phone-screened using questionnaires pertaining to inclusion and exclusion criteria and also the American Heart Association/American College of Sports Medicine exercise pre-participation screening. Prior to participation, all patients read and signed the informed consent approved by the Cleveland Clinic Institutional Review Board.

Procedures

Participants meeting the inclusion criteria were asked to make two visits to the Cleveland Clinic in Cleveland, Ohio. Visits were separated by at least 24 hours, but no more than 72 hours. On the first visit, after obtaining informed consent, participants were clinically assessed for PD by a Movement Disorders Neurologist and rated using the UPDRS Part-III Motor Exam (mean = 1.28, SD = 0.84) while “off” anti-parkinsonian medication for at least 12 hours prior to examination (UPDRS gait: mean = 0.76, SD = 0.66; UPDRS posture: mean = 0.52, SD = .59).

On the second visit, patients were asked to undergo a graded maximal oxygen uptake exercise test (VO_2) done on a cycle ergometer (Lode Excalibur Sport with Pedal Force Measurement, Lode B.V., Groningen, Netherlands), which was fitted to each participant. Patients reported to their appointment “on” their anti-parkinsonian medication and having fasted from food and drink, except water, for four hours and caffeine for at least 12 hours. Anthropometric recordings of height and weight (Seca 644, Hamburg, Germany) were recorded and medical history was taken. Patients were familiarized with the cycle ergometer, standard 12-lead electrocardiogram and ratings of perceived exertion Borg scale (RPE) to be utilized during the protocol. Electrocardiogram (Welch Allyn, Cardioperfect, Skaneateles Falls, NY) was monitored throughout the entire graded exercise test (GXT), with blood pressure and RPE taken during the last 15 seconds of every stage. VO_2 was recorded throughout the GXT via indirect calorimetry using a calibrated metabolic cart (Medgraphics, Minneapolis, MN) with each patient fitted with a mouthpiece and nose clips. Crank torque and power output per limb were measured throughout the cycle test using strain gauges built into the crank arms of the cycle ergometer (Lode Excalibur Sport with Pedal Force Measurement, Lode B.V., Groningen, Netherlands). Each enrolled patient successfully completed the protocol.

Patients began with a five-minute supine rest prior to the warm-up phase of the GXT. The warm-up phase consisted of three minutes cycling at 20W resistance at a self-selected pedaling cadence. Patients were encouraged to maintain this self-selected cadence

throughout the GXT. After warm-up, each stage lasted two minutes in length with 20W increases until stage four (minute eight) when 40W increases were made until volitional exhaustion, or until the American College of Sport Medicine test termination criteria was met [26]. Three minutes of recovery cycling at 20W was completed by each patient. This test is a modification of previously described protocols used in the PD population [27, 28].

Based on torque data measured for each limb, a Symmetry Index (SI) was calculated for each 360-degree pedal revolution. The Symmetry Index formula was applied to each pedal revolution of the GXT, using the average power (AveSI), maximum power (MaxSI), and minimum power (MinSI) generated by each limb at each revolution. A positive SI indicated a greater contribution by the unaffected limb and a negative SI indicated a greater contribution from the affected limb.

$$Symmetry \quad Index \quad (SI) = \frac{Unaffected \quad Limb - Affected \quad Limb}{(Unaffected \quad Limb + Affected \quad Limb)/2}$$

In reviewing the cycling kinetics of the PD participants, it was observed that there typically was a portion of the pedal cycle near top and bottom dead center in which the net torque produced about the crank axle was very low, much lower than that typically displayed by a healthy, recreational cyclist [29]. The duration of this low torque (LTD) was calculated for each full 360-degree pedal revolution as the number of degrees of the pedal cycle in which the net torque exerted on the pedals by both legs was below ten percent of that revolution's maximum net torque output. Higher proportions of the pedal revolution spent at this low torque level are indicative of a less smooth pedaling pattern (see Figure 1.).

Three stages of exercise were selected for further analysis, as each patient completed at minimum 3 stages of the cycle test. The first stage represents a low intensity workload, the initial 20W stage of the GXT; the second stage represents a moderate workload, 60 W; and the last stage represents a maximal intensity workload, each subject's final completed stage.

Data Analysis

All data were analyzed using MATLAB. Statistical analysis was performed using the SPSS statistical software (SPSS 18, IBM SPSS software, Armonk, NY.). Significance level for all analyses was set at $P<0.05$. A repeated measures analysis of variance was used to test for differences between the three stages of exercise and the different measures of SI (AveSI, MaxSI, MinSI) and LTD. Greenhouse Geyser adjustment was reported when the sphericity assumption was violated. Post hoc contrasts (Bonferroni adjustment) were used to determine differences between the three stages. To assess the relationship between measures of pedaling kinetics and clinical measures of gait and postural stability, bivariate Pearson correlations were calculated.

RESULTS

Pedaling cycles were averaged for each workload. Example plots of individual leg torque and net torque throughout the pedaling cycle are shown for one participant in Figure 1.

Asymmetry between affected and unaffected side lower limbs can be seen in the difference in magnitude at peak output during the pedaling cycle.

For differences between exercise stages, AveSI ($F(1.04, 24.89) = 13.32$, $p = .001$, $\eta^2 = .357$), MaxSI ($F(1.50, 35.98) = 6.78$, $p = .006$, $\eta^2 = .220$) and LTD ($F(1.38, 35.54) = 13.32$, $p < .001$, $\eta^2 = .597$) were found to exhibit a significant effect of exercise stage, whereas for MinSI, effect was not significant ($F(1.59, 38.26) = 1.62$, $p = .214$, $\eta^2 = .063$) (See Table 1.). AveSI and LTD were significantly different at all three stages of exercise (20W, 60W, maximum workload), with positive AveSI values decreasing from 20W (1.18) to maximum workload (0.13) and LTD decreasing from 20W (73.17°) to maximum workload (41.77°). MaxSI at 20W and 60W was significantly different from MaxSI at maximum workload. Also MaxSI decreased with increasing exercise load. That means the contribution of the affected limb increased with increasing exercise load in AveSI and MaxSI (see Figure 2).

For relationships between measures of pedaling kinetics and measures of gait and posture, we did not find a significant correlation between measures of pedaling kinetics (symmetry indices, torque measure) and measures of gait and posture ($r = .01$ to $r = .31$; .always $p > .05$), with one exception: MinSI at maximum revolution correlated significantly with posture ($r = .41$, $p = .04$) (see Table 2.).

DISCUSSION

Postural instability and gait dysfunction are two indicators of motor deficits in PD patients. Interventions targeted at alleviating this issue is of great importance as it affects ADL's. Characterizing pedaling in PD patients may provide insight into potential future issues with postural instability and gait dysfunction and also provide a measurement that is a more sensitive assessment of lower extremity function than the current scale, the UPDRS. Understanding pedaling and subsequent asymmetries in a PD population would provide a baseline for comparison of effectiveness of interventions targeting postural instability and gait dysfunction, especially when compared with the clinical UPDRS ratings of those symptom deficits.

Decreases in SI (symmetry increases) were observed for average power output in PD patients as workload increased from the initial 20W stage to the intermediate 60W stage and finally to the maximal exertion stage of the exercise test. Maximum power SI showed a significant difference in symmetry between performance at both the 20W and 60W stage and the maximal resistance stage. Minimum power SI did not show significant differences across the three selected stages. Average power proved to be the most asymmetrical of the three metrics, with greater SI at earlier stages when compared to maximum and minimum power (Figure 2). This finding parallels with data from healthy populations. It was hypothesized that pedaling asymmetries would be found in PD patients, to a similar degree as found in healthy populations. As expected, based on literature research on healthy norms, SI values decreased (showing increased symmetry) with increasing workload. SI values generated from maximum and minimum power values were within normal ranges of asymmetry (less than 20%).

The finding that the variability in pedaling, otherwise seen in the SD for SI, decreased from 20W to 60W to the last stage in the exercise test is of particular interest. Previous studies have shown that PD patients experience a decrease in the quantity, quality and processing of afferent information which is thought to be a result of decreased cortical activation [30, 31]. Increased variability in pedaling kinetics may contribute to the decrease in the quality and processing of afferent information. The results of this study, have shown variability in pedaling motion in individuals with PD. The findings may help to support a need for a therapeutic intervention that provides higher quality and quantity afferent information via augmenting the pedaling motion. An intervention such as forced-exercise could potentially aid in better afferent processing, leading to improvement or alleviation of postural instability and gait dysfunction and an increase in quality of life.

Based on the relationships observed with the maximum and minimum torques, the extrema of the overall torque profiles are consistent in magnitude across stages of the exercise test. At the same time, the symmetry of the average power output is increasing with increasing resistance. In investigating these phenomena, a “low torque” area of varying duration was found in the net torque profile. This LTD indicated that a larger portion of each pedaling revolution is spent exerting negative torque (i.e. pulling up on the pedal or not exerting significant torque) and that the drive lower extremity was contributing a more gradual increase in torque. This combination seemed to contribute to the “low torque area.” Increased “pull” by the leg at back end of pedaling circuit leads to increased negative torques that effectively cancels out normal or decreased positive torque by the other limb, the drive lower extremity. Especially at lower resistance stages, this LTD is highly variable in length, lending credence to the idea that it is a measure of subject engagement and symmetry. With increasing resistance, the “need” for symmetry and an efficient pedaling motion seems to curtail this LTD down to less than 12% of the pedaling cycle from over 20% at the initial stage.

The secondary hypothesis was that pedaling asymmetries that were found in individuals with PD would show positive association with clinical measures of gait and postural stability, with worsening clinical ratings corresponding to increased cycling asymmetry. The anticipated relationship between pedaling asymmetry and postural instability and gait dysfunction scores on the UPDRS was not found in this study. While cycling kinetics has not traditionally been used as a clinical tool for patients with PD, we have shown that even if asymmetries are present, the UPDRS may not be sensitive enough to detect these asymmetries. The subjective nature of the UPDRS evaluation limits the precision with which postural instability and gait dysfunction can be quantified, as seen in the SI during low resistance pedaling. Since no relationship between postural instability and gait dysfunction and SI was determined in this study despite evidence suggesting the presence of asymmetries, perhaps the UPDRS may be relatively insensitive to the effectiveness of medical or surgical intervention.

Study limitations

Our research included those with PD who were mild to moderate in their disease state. Therefore, our results may only be applicable to a subset of the PD population. We also did

not take into account if there were differences between genders. Future research expanding to PD patients with severe motor impairments, and also research studying gender differences in PD may help to clarify and improve our findings. Furthermore, patients reported to their GXT visit on their anti-parkinsonian medication and the UPDRS exam was preformed off their antiparkinsonian medication. Ideally, clinical assessments and metabolic testing would have been completed while patients were in the “on” and “off” medication states. The additional burden required of the patient to be “off” medication for additional sessions led to the decision to not collect kinetic and clinical data in all phases of medication. Future research investigating pedaling patterns compared to the UPDRS while patients are in similar medicated states may be helpful to further reveal the relationship between clinical ratings of PD and PIGD.

CONCLUSION

In summary, it was found that lower extremity asymmetries were present in PD patients during pedaling on a cycle ergometer during a graded, maximal effort exercise stress test, and that these asymmetries decreased with increases in workload. It was also found that asymmetries in pedaling did not correlate to scores of postural instability and gait as rated using the UPDRS. Future studies are needed to determine how to minimize postural instability and gait dysfunction in PD patients through therapeutic interventions and to develop more sensitive diagnostic tools for more effective measurement of postural instability and gait dysfunction in neurological populations. Demonstrating that PD patients exhibit asymmetrical pedaling kinetics provides support for an intervention, such as forced exercise, that attempts to normalize patient's movement symmetry. The characterization of pedaling kinetics in PD patients may be useful clinically for early detection of gait and postural instability caused by disease progression. Earlier detection may provide an opportunity for more effective intervention to offset the advance and severity of the imbalances.

ACKNOWLEDGEMENTS

This research was funded by NIH grant R21HD056316A. Study sponsors had no role in the study design, collection, analysis and interpretation of data; in the writing of the manuscript; and in the decision to submit the manuscript for publication.

REFERENCES

1. Deponti GN, Silva DR, Coelho AC, Muller AM, Dalcin Pde T. Delayed diagnosis and associated factors among new pulmonary tuberculosis patients diagnosed at the emergency department of a tertiary care hospital in Porto Alegre, South Brazil: a prospective patient recruitment study. BMC Infect Dis. 2013; 13:538. [PubMed: 24219185]
2. Holtbernd F, Eidelberg D. Functional brain networks in movement disorders: recent advances. Curr Opin Neurol. Aug; 2012 25(4):392–401. [PubMed: 22710361]
3. Bloem BR, Beckley DJ, van Hilten BJ, Roos RA. Clinimetrics of postural instability in Parkinson's disease. Journal of Neurology. Oct; 1998 245(10):669–73. [PubMed: 9776467]
4. Schrag A, Sampaio C, Counsell N, Poewe W. Minimal clinically important change on the unified Parkinson's disease rating scale. Mov Disord. Aug; 2006 21(8):1200–7. [PubMed: 16673410]

5. Muslimovic D, Post B, Speelman JD, Schmand B, de Haan RJ. Determinants of disability and quality of life in mild to moderate Parkinson disease. Neurology. Jun 3; 2008 70(23):2241–7. [PubMed: 18519873]
6. Schrag A, Jahanshahi M, Quinn N. What contributes to quality of life in patients with Parkinson's disease? J Neurol Neurosurg Psychiatry. Sep; 2000 69(3):308–12. [PubMed: 10945804]
7. Brown D, Kukulka C. Human Flexor Reflex Modulation During Cycling. J Neurophysiol. 1993; 69(4):1212–24. [PubMed: 8492160]
8. Ting L, Kautz S, Brown D, Zajac F. Contralateral Movement and Extensor Force Generation Alter Flexion Phase Muscle Coordination in Pedaling. J Neurophysiol. 2000; 82:3351–65. [PubMed: 10848554]
9. Yang J, Stein R. Phase-Dependent Reflex Reversal in Human Leg Muscles During Walking. J Neurophysiol. 1990; 63(5):1109–17. [PubMed: 2358865]
10. Zehr EP, Balter JE, Ferris DP, Hundza SR, Loadman PM, Stoloff RH. Neural regulation of rhythmic arm and leg movement is conserved across human locomotor tasks. J Physiol. Jul 1; 2007 582(Pt 1):209–27. [PubMed: 17463036]
11. Fowler E, Kolobe T, Damiano D, Thorpe D, Morgan D, Brunstrom J, Coster W, Henderson R, Pitetti K, Rimmer J, Rose J, Stevenson R. Promotion of Physical Fitness and Prevention of Secondary Conditions for Children with Cerebral Palsy: Section on Pediatrics Research Summit Proceedings. Phys Ther. 2007; 87(11):1495–510. [PubMed: 17895351]
12. Lennon O, Carey A, Gaffney N, Stephenson J. A pilot randomized controlled trial to evaluate the benefit of cardiac rehabilitation paradigm for the non-acute ischmaemic stroke population. Clin Rehabil. 2008; 22:125–33. [PubMed: 18212034]
13. Pontichtera-Mulcare J. Exercise and multiple sclerosis. Med Sci Sports Exerc. 1992:451–65.
14. Quaney B, Boyd L, McDowd J, Zahner L, He J, Mayo M, Macko R. Aerobic exercise improves cognition and motor function poststroke. Neurorehabil Neural Repair. 2009; 23(9):879–85. [PubMed: 19541916]
15. Studenski S, Duncan PW, Perera S, Reker D, Lai SM, Richards L. Daily functioning and quality of life in a randomized controlled trial of therapeutic exercise for subacute stroke survivors. Stroke. Aug; 2005 36(8):1764–70. [PubMed: 16040590]
16. Bini RR, Diefenthaeler F, Mota CB. Fatigue effects on the coordinative pattern during cycling: kinetics and kinematics evaluation. J Electromyogr Kinesiol. Feb; 2010 20(1):102–7. [PubMed: 19028111]
17. Carpes F, Rossato M, Faria I, Mota CB. During an incremental exercise cyclists improve bilateral pedaling symmetry. Brazilian journal of biomotricity. 2008:155–9.
18. Ericson M, Bratt A, Nisell R, Arborelius U, Ekholm J. Power output and work in different muscle groups during ergometer cycling. Eur J Appl Physiol. 1986; 55:229–35.
19. Lee H, Martin D, Anson J, Grundy D, Hahn A. Physiological characteristics of successful mountain bikers and professional road cyclists. J Sports Sci. 2001; 20(12):1001–8. [PubMed: 12477010]
20. Too D, Landwer G. The effect of pedal crank arm length on joint angle and power production in upright cycle ergometry. J Sports Sci. 2001; 18(3):153–61. [PubMed: 10737266]
21. Ridgel AL, Vitek JL, Alberts JL. Forced, not voluntary, exercise improves motor function in Parkinson's disease patients. Neurorehabilitation and Neural Repair. Jul-Aug;2009 23(6):600–8. [PubMed: 19131578]
22. Vautier J, Vandewalle H, Arabi H, Monod H. Critical power as an endurance index. Appl Ergon. 1995; 26(2):117–21. [PubMed: 15677008]
23. Sanderson DJ. The influence of cadence and power output on asymmetry of force application during steady-rate cycling. Journal of human movement studies. 1990; 19:1–9.
24. Daly D, Cavanagh P. Asymmetry in bicycle ergometer pedaling. Med Sci Sports Exer. 1976; 8(3): 204–8.
25. Smak W, Neptune R, Hull M. The influence of pedaling rate on bilateral asymmetry in cycling. J Biomech. 1999; 32:899–906. [PubMed: 10460126]
26. Thompson, WR. Medicine ACoS. In: Gordon, NF.; Pescatello, LS., editors. ACSM's Guidelines for Exercise Testing and Prescription. 8 ed. Lippincott Williams & Wilkins; 2009.

27. Canning C, Alison J, Allen N, Groeller H. Parkinson's Disease: An Investigation of Exercise Capacity, Respiratory Function, and Gait. Archives of Physical Medicine and Rehabilitation. 1997; 78:199–207. [PubMed: 9041903]

28. Stanley RK, Protas EJ, Jankovic J. Exercise performance in those having Parkinson's disease and healthy normals. Med Sci Sports Exerc. Jun; 1999 31(6):761–6. [PubMed: 10378900]

29. Faria, I.; Cavanagh, PR. The physiology and biomechanics of cycling. Wiley; New York: 1978.

30. MR D. Primate models of movement disorders of basal ganglia origin. Trends in Neuroscience. 1990; 13(7):281–5.

31. Turner R. The functional anatomy of parkinsonian bradykinesia. Neuroimage. 2003

Highlights

- Pedaling kinetics were characterized in a Parkinson's disease population
- The relationship between pedaling kinetics and clinical measures of gait and postural stability were assessed
- Asymmetries in pedaling kinetics of PD patients were found
- Pedaling asymmetries were not correlated to clinical measures of gait and postural instability scores

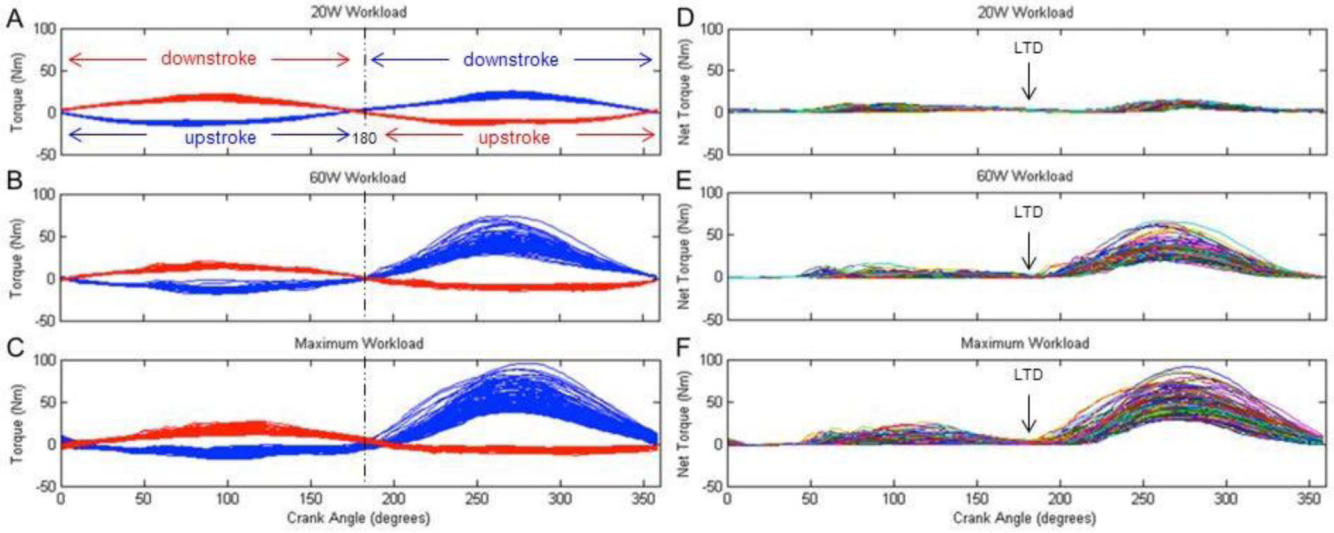

Figure 1.

Example plots of one participant's individual leg torque profiles (A-C; Affected Side in red and Unaffected Side in blue) and Net Torque profiles (D-F) for all pedal revolutions during 20W (A & D), 60W (B & E), and Maximum (C & F) Workloads. The area of low torque duration (LTD) is also displayed in the Net Torque profiles.

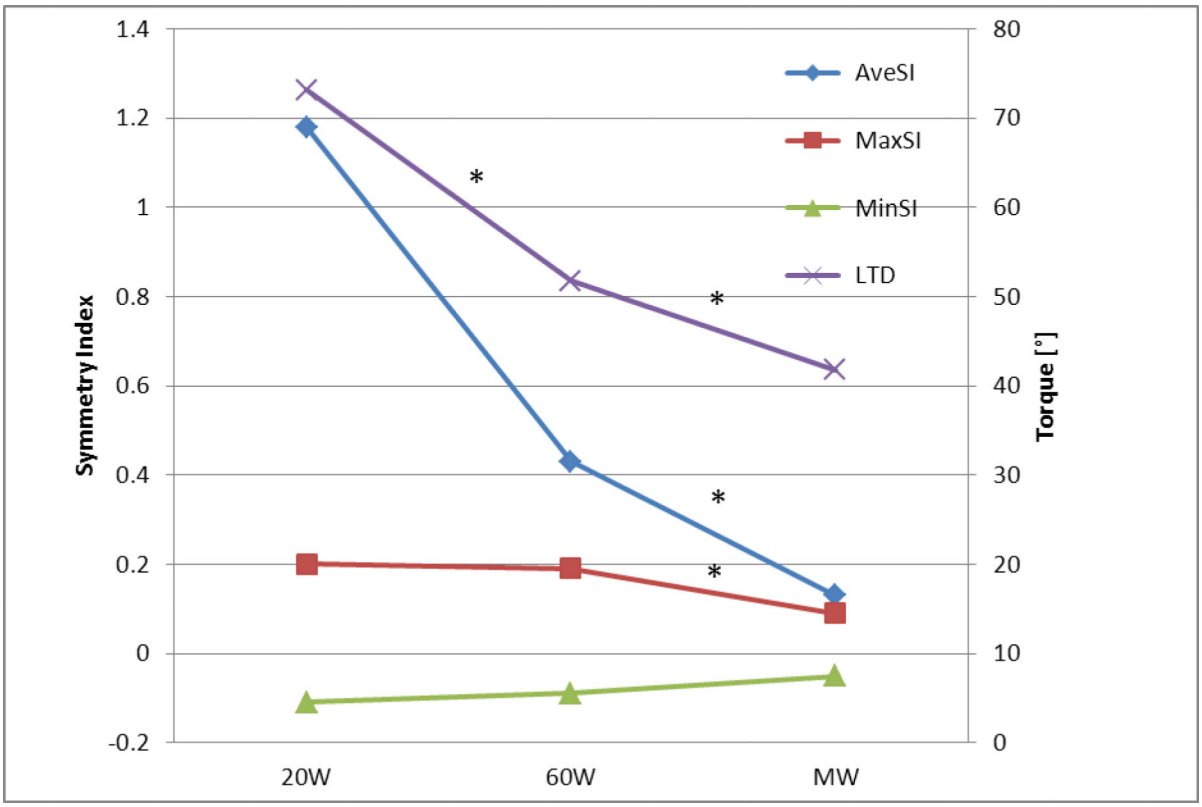

Figure 2.

Results of the symmetry indices (average power (AveSI), maximum power (MaxSI), and minimum power (MinSI)) and the torque profile (duration of low torque (LTD)) at 20W, 60W and maximum workload (MW) (for SD see Table 1). A star marks significant changes ($p < .05$), a star (*) marks marginally significant changes ($p < .10$) between workloads.

Table 1

Participant demographics

Participant	Age (yrs)	Disease Duration (yrs)	VO_{2max} (ml/kg/min)	Maximal Exercise Stage (W)	UPDRS Total Score	UPDRS Posture Score	UPDRS Gait Score
1	52	6	31.6	220	9	0	0
2	58	9	15	80	31	1	1
3	59	6	21.6	100	20	0	0
4	44	8	29.4	140	34	2	1
5	67	5	21.9	140	27.5	0	1
6	72	5	12.4	80	6	0	0
7	61	5	21.9	180	13	2	1
8	56	1	16.5	80	31	1	1
9	56	2	28.1	180	15	1	0
10	70	0.5	26.2	180	18	1	0
11	50	3	27.1	140	15	0	1
12	53	0.5	20.6	180	14	0	0
13	71	3	20.4	100	27	0	2
14	47	1	25.1	220	19	0	1
15	57	2	25.7	140	14	0	1
16	71	2	18.7	140	37	1	0
17	53	4	18.7	100	24	1	1
18	65	0.5	17.6	80	16	1	0
19	67	9	22.5	80	24	1	2
20	51	4.5	12.5	80	15	0	2
21	67	1	20.4	180	23	1	0
22	66	10	15.9	80	25	0	1
23	45	1.5	30.9	260	35	0	1
24	58	0.5	20.6	80	15	0	0
25	60	0.5	22.4	100	22	1	1
Mean and SD	59.04 ± 8.45	3.62 ± 3.01	21.75 ± 5.32	133.6±53.76	21.18 ± 8.30	.56 ± .65	.72 ± .68

Table 2

Pearson product moment correlations for clinical measures (UPDRS_total (UPDRS), gait, posture) and pedaling kinetics (AveSI, MaxSI, MinSI, LTD) at each revolution (20W, 60W, maxW).

Variables	UPDRS	gait	posture
1. AveSI_20W	−.18	.02	−.27
2. AveSI_60W	−.06	.06*	−.15
3. AveSI_max	−.06	−.16	.10
1. MaxSI_20W	−.12	.03	−.21
2. MaxSI_60W	−.07	.01	−.10
3. MaxSI_max	.05	−.10	.19
1. MinSI_20W	.11	−.08	.26
2. MinSI_60W	−.17	−.29	.08
3. MinSI_max	.31	.02	.41*
1. LTD_20W	.14	.05−	.14
2. LTD_60W	.10	.07	.07
3. LTD_max	.17	.04	.21

Note.

* $p < .05$